He has seen a number of cases in which three or four injections of one or the other of these salts caused the tubercle bacilli to disappear from the sputum and the feces, and the cessation of the most profound toxemia. He reports in detail a case diagnosticated as acute miliary tuberculosis that terminated in an apparent cure. In less than eight weeks after the onset of the disease, at a time when 95 per cent. of these patients are either dead or dying, and six weeks after the administration of the first injection, the patient was apparently cured. Wright thinks that this case warrants the belief that in mercury we have the chemical affinity of the tubercle bacillus, and the hope that experimentation will sooner or later develop a compound of mercury for which these organisms will have even a stronger affinity than they display for the succinimide.

PEDIATRICS.

UNDER THE CHARGE OF

LOUIS STARR, M.D., AND THOMPSON S. WESTCOTT, M.D.,

OF PHILADELPHIA.

Purpura in Childhood with Fatal Cerebral Hemorrhage.—FEDOR SCHMEY (*Deutsch. med. Woch.*, 1911, xxxvii, 307) reports a case of purpura in a child, aged eight years, with fatal termination from cerebral hemorrhage. Cerebral hemorrhage in purpura is very rare, such an authority as Henoch having reported but one case, which developed convulsions, stupor, and hemiplegia. Schmey's case occurred in a well-developed and apparently healthy boy. When seen on October 10, the boy had been spitting up blood for several preceding mornings, and the cervical glands on both sides were swollen, but not tender. There was harsh breathing over the apex of the right lung. Physical signs were wanting to show that the blood came from the lung. There had been recent bleeding from the gums. There was prostration, but no pain. The temperature remained normal, and there was no swelling or tenderness of the joints. On October 12 there was vomiting of dark blood mixed with stomach contents. On October 15 there appeared on both legs and on the right arm about twelve dark patches the size of a penny. These patches were clear, black, round, did not disappear on pressure, and had a central elevation. On October 18 a sudden coma developed. The pupils were dilated and did not react, and the reflexes of the conjunctiva and skin were absent. The liver was enlarged to the level of the umbilicus; the pulse was slowed, but regular; and the gums were covered with small hemorrhagic areas. The temperature was 39.5° C. The coma continued until death occurred, four hours later. Convulsions did not appear, either preceding or during the coma. Shortly before death there occurred a severe hemorrhage from the mouth. These symptoms indicate a fatal cerebral hemorrhage and an extravasation into the brain substance. This case seems to stand between a purpura abdominalis and a purpura hemorrhagica, and does not conform entirely with any of the four forms as laid down by Henoch. It also shows that purpura is not a disease sui generis, but a number of processes combined in an uncertain manner, all having the tendency to produce bleeding from the skin, mucous membrane, or the parenchyma of organs. Infectious diseases seem to have a bearing on the development of purpura. The case just described had had scarlet fever two years before. Henoch's case had scarlet fever four years before the purpura. Schmey places more weight upon the fact that the child suffered from a scrofulosis, probably tuberculous. Wolf lays stress on an inherited tuberculous taint in the etiology of his cases. He also observed that bleeding began in the erect position, but disappeared in the recumbent. In Schmey's case the bleeding developed seriously after the child had been in bed for several days.

The Recognition of Eosinophilic, Intestinal Crises in Infants.—LEO LANGSTEIN (*Münch. med. Woch.*, 1911, lviii, 623) some years ago reported a condition in infants in which there occurred sudden discharges from the bowel of mucus and pus in which were found large numbers of eosinophile cells. In the absence of an alimentary or infectious bowel disturbance this condition was thought to be a partial manifestation of that symptom complex which Czerny has named an "exudative diathesis." In both of Langstein's cases symptoms of this condition were present. Czerny and Goeppert have expressed their belief that the mucous membrane of the intestine frequently shares in the manifestation of the exudative diathesis. Since then, Langstein had made a study of mucopurulent stools in infants to determine the frequency of eosinophile cells, and found the condition much more frequent than was supposed. He believes this is one of the early symptoms of the exudative diathesis. He describes a typical case in a baby aged five weeks, which developed facial eczema, and a few days later sudden copious, mucopurulent stools containing eosinophile cells in great numbers. These stools continued for two more days and ceased as suddenly as they began. The eczema became worse during this time, and the body weight diminished only slightly. There was no rise in temperature, and the blood showed an eosinophilia of 8 per cent. In this case, alimentary and infectious disturbance of the bowels being ruled out, and the view taken that the condition was a manifestation of an exudative diathesis, as shown by the eosinophile cells and the stools, no change was made in the infant's nourishment except a slight reduction in quantity. The presence of the eosinophile cells, therefore, is not only of diagnostic value, but is a guide to our regulation of the nourishment. In these cases it is unnecessary to employ a starvation diet, as in infectious and alimentary bowel disorders; and fever and loss of weight are not found as in the last two conditions.

Neurasthenia in Older Children.—CLIVE RIVIERE (*The Practitioner*, 1911, lxxxvi, 46) accepts Savill's division of neurasthenia in older children into the etiological factors of toxic malnutrition and fatigue neurasthenia, and that due to shock and traumatism. As a rule, the causation is mixed. Neurasthenia in older children is usually